Updated Dialog search
12/16/2009
(medline, embaes, scisearch, biosis)

Set	Items	Description
S1	10	HBV (S) PRES1 (S) ANTIBOD? (S) (INTERFERON OR IFN)
S2	4	RD (unique items)
S3	5	S1 NOT PY>2003

Display 3/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14708292 PMID: 11900226
Inducing oral immune regulation of hepatitis B virus envelope proteins suppresses the growth of hepatocellular carcinoma in mice.
Gotsman Israel; Alper Ruslana; Klein Athalia; Rabbani Elazar; Engelhardt Dean; Ilan Yaron
Department of Medicine, Hadassah-Hebrew University Medical Center, Jerusalem, Israel.
Cancer (United States) Jan 15 2002, 94 (2) p406-14, ISSN 0008-543X --Print Journal Code: 0374236
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
BACKGROUND: Hepatitis B virus (HBV)-associated hepatocellular carcinoma (HCC) expresses hepatitis B surface antigen (HBsAg) on its cell surface,

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Display 3/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

and this may serve as a tumor-associated antigen. It was shown previously that adoptive transfer of immunity against HBsAg facilitates the suppression of experimental human HCC-expressing HBsAg in athymic mice. The authors recently showed that it was possible to augment the anti-HBV immune response through induction of oral immune regulation for HBV-associated antigens. The objective of this study was to evaluate the effect of oral immune regulation for HBV antigens on the growth of HBsAg-expressing HCC. METHODS: Recipient athymic Balb/c mice were irradiated sublethally and injected with 10(7) human hepatoma cells followed by the adoptive transfer of 2 x 10(6) splenocytes from donor mice. Four groups of donor Balb/c mice were studied: Two groups were immune modulated through oral administration of HBV antigens (HBsAg, PreS1, and Pre S2) or bovine serum albumin (BSA). Two control groups were immunized for HBsAg and fed HBV antigens or BSA. Recipient mice were followed for tumor volume and serum alpha-fetoprotein (aFP) levels. The humoral immune response was determined by measuring serum HBs antibodies. HBV specific T-cell immune modulation was assessed in vitro by HBV specific T-cell proliferation and interferon gamma (IFNgamma)

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Display 3/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

ELISPOT assays as well as cytokine expression by reverse transcriptase-polymerse chain reaction assays. RESULTS: The adoptive transfer of orally immune modulated HBV splenocytes induced complete tumor suppression in recipient mice compared with control mice transplanted with nonimmune modulated cells (BSA), which showed significant tumor growth (serum aFP levels were 3.5 ng/mL and 2320.0 ng/mL, respectively). Control mice transplanted with anti-HBs immunized cells (with or without oral immune modulation) manifested similar tumor suppression (3.5 ng/mL and 0.5 ng/mL, respectively). Immunoregulation for HBV antigens augmented the HBV specific T-cell immune response, as manifested by an increase in HBV specific T-cell proliferation and IFNgamma ELISPOT assays in mice orally immune regulated with HBV proteins compared with naive mice. Tumor suppression was mediated through increased IFNgamma production in immune regulated and immunized mice. CONCLUSIONS: The induction of oral immune regulation for HBV antigens modulated the antitumor immune response, thus suppressing the growth of HCC in mice. This effect was mediated by the enhancement of anti-HBV specific T-cell immunity.

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Display 3/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10524652 PMID: 1280500
PreS antigen expression and anti-preS response in hepatitis B virus infections: relationship to serum HBV-DNA, intrahepatic HBcAg, liver damage and specific T-cell response.
Petit M A; Capel F; Zoulim F; Dubanchet S; Chemin I; Penna A; Ferrari C; Trepo C
INSERM Unite 131, Clamart, France.
Archives of virology. Supplementum (AUSTRIA) 1992, 4 p105-12, ISSN 0939-1983--Print Journal Code: 9214275
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The diagnostic value of preS antigens and anti-preS antibodies during Hepatitis B virus (HBV) infections have not yet been clearly elucidated.

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DIALOG(R)File 155:MEDLINE(R)

Therefore, the objectives of this study were: 1) to better understand the clinical significance of the expression of both preS1 and preS2 antigens (preS1Ag and preS2Ag) in the serum of chronic HBsAg carriers, and 2) to define the respective role of antibody responses to HBs-, preS2- and preS1-specific determinants in the course of acute hepatitis B (AH-B) infections with different outcomes. Our data showed that the serum preS1Ag/HBsAg ratio correlated well with the level of viral replication (serum HBV-DNA as monitored by PCR assay and liver HBcAg), especially in

anti-HBe positive chronic carriers. The complete eradication of virions required a persistent antibody response to conformation-dependent HBs-epitopes, temporally associated with a vigorous T cell response to nucleocapsid antigens. Recovery from hepatitis B can be achieved when there is no early antibody response to preS2- and preS1-proteins, which was found to be transient, concomitant with a flare-up of the liver disease, and preceding anti-HBs production. Information on the patterns of preS antigens and their antibodies remained clouded because of the varying specificities and sensitivities of research methods used in studies to date. We have,

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Display 3/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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therefore, developed original Polyclonal-Monoclonal RadioImmunoAssays (PAb-MAb RIAs) by using monoclonal antibodies (MAbs) having previously well-defined specificities. We could thus detect and quantify simultaneously the three distinct antigenicities of the HBV envelope, HBsAg, preS2Ag and preS1Ag, with the same sensitivity. In this way, the preS1Ag/HBsAg and preS2Ag/HBsAg ratios can be calculated to estimate the serum expression of both preS1Ag and preS2Ag in relation to total HBsAg activity during different stages of chronic HBV infection. For optimal management of the state of HBV replication in chronic viral infection, the detection of HBV-DNA in serum was monitored by the Polymerase Chain Reaction (PCR) assay. We extended our work by investigating the clinical significance of antibody response to preS-specific determinants in patients with AH-B showing different outcomes in both natural course or response to alpha-interferon therapy. In a first attempt, we chose to use the Western Immuno-Blotting Assay (WIBA) to obtain a qualitative assessment of the nature of preS antibody responses. Finally, the cell-mediated immune response to HBV antigens was also studied in several patients with

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DIALOG(R)File 155:MEDLINE(R)
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self-limited AH-B leading to a relevant finding which may help to clarify the mechanisms responsible for complete clearance of HBV.

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Display 3/3,AB/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2009 Elsevier B.V. All rts. reserv.

0078870800 EMBASE No: 2002034443
Inducing oral immune regulation of hepatitis B virus envelope proteins suppresses the growth of hepatocellular carcinoma in mice
Gotsman I.; Alper R.; Klein A.; Rabbani E.; Engelhardt D.; Ilan Y.
Liver Unit, Division of Medicine, Hadassah University Hospital, P.O. Box 12000, Jerusalem, IL-91120, Israel
CORRESP. AUTHOR/AFFIL: Ilan Y.: Liver Unit, Division of Medicine, Hadassah University Hospital, P.O. Box 12000, Jerusalem, IL-91120, Israel
CORRESP. AUTHOR EMAIL: ilan@hadassah.org.il

Cancer (Cancer) (United States) January 15, 2002, 94/2 (406-414)
CODEN: CANCA ISSN: 0008-543X
DOI: 10.1002/cncr.10237
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 55

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Display 3/3,AB/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

BACKGROUND. Hepatitis B virus (HBV)-associated hepatocellular carcinoma (HCC) expresses hepatitis B surface antigen (HBsAg) on its cell surface, and this may serve as a tumor-associated antigen. It was shown previously that adoptive transfer of immunity against HBsAg facilitates the suppression of experimental human HCC-expressing HBsAg in athymic mice. The authors recently showed that it was possible to augment the anti-HBV immune response through induction of oral immune regulation for HBV-associated antigens. The objective of this study was to evaluate the effect of oral immune regulation for HBV antigens on the growth of HBsAg-expressing HCC. METHODS, Recipient athymic Balb/c mice were irradiated sublethally and injected with 10 SUP 7 human hepatoma cells followed by the adoptive transfer of 2 x 10 SUP 6 splenocytes from donor mice. Four groups of donor Balb/c mice were studied: Two groups were immune modulated through oral administration of HBV antigens (HBsAg, PreS1, and Pre S2) or bovine serum albumin (BSA). Two control groups were immunized for HBsAg and fed HBV antigens or BSA. Recipient mice were followed for tumor volume and serum

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Display 3/3,AB/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

alpha-fetoprotein (alphaFP) levels. The humoral immune response was determined by measuring serum HBs antibodies. HBV specific T-cell immune modulation was assessed in vitro by HBV specific T-cell proliferation and interferon gamma (IFN?) ELISPOT assays as well as cytokine expression by reverse transcriptase-polymerse chain reaction assays. RESULTS. The adoptive transfer of orally immune modulated HBV splenocytes induced complete tumor suppression in recipient mice compared with control mice transplanted with nonimmune modulated cells (BSA), which showed significant tumor growth (serum alphaFP levels were 3.5 ng/mL and 2320.0 ng/mL, respectively). Control mice transplanted with anti-HBs immunized cells (with or without oral immune modulation) manifested similar tumor suppression (3.5 ng/mL and 0.5 ng/mL, respectively). Immunoregulation for HBV antigens augmented the HBV specific T-cell immune response, as manifested by an increase in HBV specific T-cell proliferation and IFNgamma ELISPOT assays in mice orally immune regulated with HBV proteins compared with naigammave mice. Tumor suppression was mediated through increased IFNgamma production in immune regulated and immunized mice. CONCLUSIONS.

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Display 3/3,AB/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

The induction of oral immune regulation for HBV antigens modulated the antitumor immune response, thus suppressing the growth of HCC in mice. This effect was mediated by the enhancement of anti-HBV specific T-cell immunity. (c) 2002 American Cancer Society.

- end of record -

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Display 3/3,AB/4 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

16588751 BIOSIS NO.: 200200182262
Inducing oral immune regulation of hepatitis B virus envelope proteins suppresses the growth of hepatocellular carcinoma in mice
AUTHOR: Gotsman Israel; Alper Ruslana; Klein Athalia; Rabbani Elazar; Engelhardt Dean; Ilan Yaron (Reprint)
AUTHOR ADDRESS: Liver Unit, Division of Medicine, Hadassah University Hospital, Jerusalem, IL-91120, Israel**Israel
JOURNAL: Cancer 94 (2): p406-414 January 15, 2002 2002
MEDIUM: print
ISSN: 0008-543X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: BACKGROUND: Hepatitis B virus (HBV)-associated hepatocellular carcinoma (HCC) expresses hepatitis B surface antigen (HBsAg) on its cell

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Display 3/3,AB/4 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

surface, and this may serve as a tumor-associated antigen. It was shown previously that adoptive transfer of immunity against HBsAg facilitates the suppression of experimental human HCC-expressing HBsAg in athymic mice. The authors recently showed that it was possible to augment the anti-HBV immune response through induction of oral immune regulation for HBV-associated antigens. The objective of this study was to evaluate the effect of oral immune regulation for HBV antigens on the growth of HBsAg-expressing HCC. METHODS: Recipient athymic Balb/c mice were irradiated sublethally and injected with 107 human hepatoma cells followed by the adoptive transfer of 2X106 splenocytes from donor mice. Four groups of donor Balb/c mice were studied: Two groups were immune modulated through oral administration of HBV antigens (HBsAg, PreS1, and PreS2) or bovine serum albumin (BSA). Two control groups were immunized for HBsAg and fed HBV antigens or BSA. Recipient mice were followed for tumor volume and serum alpha-fetoprotein (alphaFP) levels. The humoral immune response was determined by measuring serum HBs antibodies. HBV specific T-cell immune modulation was assessed in vitro by HBV specific

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Display 3/3,AB/4 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

T-cell proliferation and interferon gamma (IFNgamma) ELISPOT assays as well as cytokine expression by reverse transcriptase-polymerse chain reaction assays. RESULTS: The adoptive transfer of orally immune modulated HBV splenocytes induced complete tumor suppression in recipient mice compared with control mice transplanted with nonimmune modulated cells (BSA), which showed significant tumor growth (serum alphaFP levels were 3.5 ng/mL and 2320.0 ng/mL, respectively). Control mice transplanted with anti-HBs immunized cells (with or without oral immune modulation) manifested similar tumor suppression (3.5 ng/mL and 0.5 ng/mL, respectively). Immunoregulation for HBV antigens augmented the HBV specific T-cell immune response, as manifested by an increase in HBV specific T-cell proliferation and IFNgamma ELISPOT assays in mice orally immune regulated with HBV proteins compared with naive mice. Tumor suppression was mediated through increased IFNgamma production in immune regulated and immunized mice. CONCLUSIONS: The induction of oral immune regulation for HBV antigens modulated the antitumor immune response, thus suppressing the growth of HCC in mice. This effect was mediated by the

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Display 3/3,AB/4 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

enhancement of anti-HBV specific T-cell immunity.

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Display 3/3,AB/5 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

10304871 Genuine Article#: 512CP Number of References: 55
Title: Inducing oral immune regulation of hepatitis B virus envelope proteins suppresses the growth of hepatocellular carcinoma in mice (ABSTRACT AVAILABLE)
Author: Gotsman I; Alper R; Klein A; Rabbani E; Engelhardt D; Ilan Y (REPRINT)
Corporate Source: Hadassah Univ Hosp,Div Med, Liver Unit,POB 12000/IL-91120 Jerusalem//Israel/ (REPRINT); Hadassah Univ Hosp,Div Med, Liver Unit,IL-91120 Jerusalem//Israel/; ENZO Biochem New York,Syosset//NY/
Journal: CANCER, 2002, V94, N2 (JAN 15), P406-414
ISSN: 0008-543X Publication Date: 20020115
Publisher: JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012 USA
Language: English Document Type: ARTICLE
Abstract: BACKGROUND, Hepatitis B virus (HBV)-associated hepatocellular carcinoma (HCC) expresses hepatitis B surface antigen (HBsAg) on its

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Display 3/3,AB/5 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

cell surface, and this may serve as a tumor-associated antigen. It was shown previously that adoptive transfer of immunity against HBsAg facilitates the suppression of experimental human HCC-expressing HBsAg in athymic mice. The authors recently showed that it was possible to

augment the anti-HBV immune response through induction of oral immune regulation for HBV-associated antigens. The objective of this study was to evaluate the effect of oral immune regulation for HBV antigens on the growth of HBsAg-expressing HCC.

METHODS. Recipient athymic Balb/c mice were irradiated sublethally and injected with 10(7) human hepatoma cells followed by the adoptive transfer of 2 x 10(6) splenocytes from donor mice. Four groups of donor Balb/c mice were studied: Two groups were immune modulated through oral administration of HBV antigens (HBsAg, PreS1, and PreS2) or bovine serum albumin (BSA). Two control groups were immunized for HBsAg and fed HBV antigens or BSA. Recipient mice were followed for tumor volume and serum alpha-fetoprotein (alphaFP) levels. The humoral immune

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Display 3/3,AB/5 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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response was determined by measuring serum HBs antibodies, HBV specific T-cell immune modulation was assessed in vitro by HBV specific T-cell proliferation and interferon gamma (IFNgamma) ELISPOT assays as well as cytokine expression by reverse transcriptase-polymerse chain reaction assays.

RESULTS. The adoptive transfer of orally immune modulated HBV splenocytes induced complete tumor suppression in recipient mice compared with control mice transplanted with nonimmune modulated cells (BSA), which showed significant tumor growth (serum aFP levels were 3.5 ng/mL and 2320.0 ng/mL, respectively). Control mice transplanted with anti-HBs immunized cells (with or without oral immune modulation) manifested similar tumor suppression (3,5 ng/mL and 0.5 ng/mL, respectively). Immunoregulation for HBV antigens augmented the HBV specific T-cell immune response, as manifested by ail increase in HBV specific T-cell proliferation and IFNgamma ELISPOT assays in mice orally immune regulated with HBV proteins compared with naive mice.

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Display 3/3,AB/5 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

Tumor suppression was mediated through increased IFNgamma production in immune regulated and immunized mice.

CONCLUSIONS, The induction of oral immune regulation for HBV antigens modulated the antitumor immune response, thus suppressing the growth of HCC in mice. This effect was mediated by the enhancement of anti-HBV specific T-cell immunity. (C) 2002 American Cancer Society.

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Set	Items	Description
S1	267	AU='WEI JUN':AU='WEI JUN-CHAO'
S2	6	S1 AND HBV
S3	4	RD (unique items)

Display 3/8/1 (Item 1 from file: 155)
DIALOG(R)File 155:(c) format only 2009 Dialog. All rts. reserv.

16491019 PMID: 15810073
Identification of the immunogenic domains in HBsAg preS1 region using overlapping preS1 fragment fusion proteins.
Apr 14 2005
Tags: Female
Descriptors: *Hepatitis B--immunology--IM; *Hepatitis B Antibodies --immunology--IM; *Hepatitis B Surface Antigens--immunology--IM; *Hepatitis B Vaccines--immunology--IM; *Hepatitis B virus--immunology--IM; *Protein Precursors--immunology--IM; Animals; Epitopes; Epitopes, B-Lymphocyte --immunology--IM; Hepatitis B--prevention and control--PC; Hepatitis B Antibodies--blood--BL; Hepatitis B Surface Antigens--chemistry--CH; Humans; Immunization; Mice; Mice, Inbred BALB C; Protein Precursors--chemistry--CH; Protein Structure, Tertiary; Viral Fusion Proteins--chemistry--CH; Viral Fusion Proteins--immunology--IM
CAS Registry No.: 0 (Epitopes); 0 (Epitopes, B-Lymphocyte); 0 (Hepatitis B Antibodies); 0 (Hepatitis B Surface Antigens); 0 (Hepatitis B Vaccines); 0 (Protein Precursors); 0 (Viral Fusion

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Display 3/8/1 (Item 1 from file: 155)
DIALOG(R)File 155:(c) format only 2009 Dialog. All rts. reserv.
Proteins); 0 (presurface protein 1, hepatitis B surface antigen)

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Display 3/8/2 (Item 2 from file: 155)
DIALOG(R)File 155:(c) format only 2009 Dialog. All rts. reserv.

15979943 PMID: 15188054
Expression of overlapping PreS1 fragment recombinant proteins for the determination of immunogenic domains in HBsAg PreS1 region.
Jun 2004
Tags: Female
Descriptors: *Hepatitis B Surface Antigens--chemistry--CH; *Recombinant Proteins--chemistry--CH; Animals; Binding, Competitive; Blotting, Western; Cloning, Molecular; DNA Primers--chemistry--CH; Enzyme-Linked Immunosorbent Assay; Escherichia coli--metabolism--ME; Glutathione Transferase --metabolism--ME; Hepatitis B virus--metabolism--ME; Mice; Mice, Inbred BALB C; Plasmids--metabolism--ME; Polymerase Chain Reaction; Protein Structure, Secondary; Protein Structure, Tertiary; Recombinant Fusion Proteins--chemistry--CH; Recombinant Fusion Proteins--metabolism--ME
CAS Registry No.: 0 (DNA Primers); 0 (Hepatitis B Surface Antigens); 0 (Recombinant Fusion Proteins); 0 (Recombinant Proteins)
Enzyme No.: EC 2.5.1.18 (Glutathione Transferase)

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Display 3/8/3 (Item 3 from file: 155)
DIALOG(R)File 155:(c) format only 2009 Dialog. All rts. reserv.

14726574 PMID: 11925607
Detection of anti-preS1 antibodies for recovery of hepatitis B patients

by immunoassay.
Apr 2002
  Descriptors: *Hepatitis B Surface Antigens--immunology--IM; *Immunoassay --methods--MT; *Protein Precursors--immunology--IM; *Viral Envelope Proteins--immunology--IM; Amino Acid Sequence; Antibodies--blood--BL; Base Sequence; Genetic Vectors; Hepatitis B--blood--BL; Hepatitis B--immunology --IM; Hepatitis B Surface Antigens--genetics--GE; Humans; Molecular Sequence Data; Peptide Fragments--immunology--IM; Peptide Fragments --metabolism--ME; Protein Precursors--genetics--GE; Recombinant Fusion Proteins--genetics--GE; Recombinant Fusion Proteins--immunology--IM; Recombinant Fusion Proteins--metabolism--ME; Viral Envelope Proteins --genetics--GE
  CAS Registry No.: 0 (Antibodies); 0 (Hepatitis B Surface Antigens); 0 (Peptide Fragments); 0 (Protein Precursors); 0 (Recombinant Fusion Proteins); 0 (Viral Envelope Proteins); 0 (presurface protein 1,

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      Display 3/8/3   (Item 3 from file: 155)
DIALOG(R)File 155:(c) format only 2009 Dialog. All rts. reserv.
hepatitis B surface antigen)

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      Display 3/8/4   (Item 4 from file: 155)
DIALOG(R)File 155:(c) format only 2009 Dialog. All rts. reserv.

14654119 PMID: 11814471
  Development of the diagnostic immunoassay to detect anti-PreS1(21-47aa) antibody--a marker suggesting the health improvement of hepatitis B patients.
Mar 2002
  Descriptors: *Antibodies--blood--BL; *Enzyme-Linked Immunosorbent Assay --methods--MT; *Hepatitis B--immunology--IM; *Hepatitis B Surface Antigens --immunology--IM; *Protein Precursors--immunology--IM; Amino Acid Sequence; Base Sequence; Epitopes; Escherichia coli--genetics--GE; Follow-Up Studies; Hepatitis B--drug therapy--DT; Hepatitis B Surface Antigens--genetics--GE; Humans; Molecular Sequence Data; Plasmids; Protein Precursors--genetics--GE ; Recombinant Fusion Proteins--genetics--GE; Recombinant Fusion Proteins --immunology--IM; Recombinant Fusion Proteins--isolation and purification --IP; Reproducibility of Results
  CAS Registry No.: 0 (Antibodies); 0 (Epitopes); 0 (Hepatitis B Surface Antigens); 0 (Protein Precursors); 0 (Recombinant Fusion Proteins); 0 (presurface protein 1, hepatitis B surface antigen)